# Intraoperative direct measurement of left ventricular outflow tract gradients to guide surgical myectomy for hypertrophic cardiomyopathy

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**Objectives:** We sought to summarize our recent experience with intraoperative monitoring for management of patients undergoing surgical myectomy for hypertrophic obstructive cardiomyopathy with emphasis on dynamic left ventricular outflow tract obstruction. We also analyzed the impact of these data on surgical decision-making and adequacy of septal myectomy.

**Methods:** We retrospectively analyzed the medical records of 198 patients who underwent transaortic septal myectomy and evaluated baseline and provoked left ventricular outflow tract gradients obtained by Doppler echocardiography and by direct measurement of pressures in the left ventricle and aorta.

**Results:** After induction of anesthesia before myectomy, left ventricular outflow tract obstruction, assessed by direct measurement, was less than the gradient documented by preoperative Doppler echocardiography in 119 patients (60%) (41 ± 31 vs 76 ± 40 mm Hg; $P < .001$). In 75 patients (38%), the obstruction was more severe (64 ± 32 vs 35 ± 31 mm Hg; $P < .001$); 4 patients (2%) had similar left ventricular outflow tract gradients. After myectomy, left ventricular outflow tract gradient decreased markedly (49 ± 33 vs 4 ± 8 mm Hg [$P < .001$] by direct measurement; 59 ± 42 vs 4 ± 6 mm Hg [$P < .001$] by transesophageal echocardiography). Cardiopulmonary bypass was resumed for more extensive myectomy in 8 (4%) patients because of a persistent residual left ventricular outflow tract gradient of 33 ± 14 mm Hg. Of note, for 78 patients (39%) intraoperative Doppler echocardiographic assessment of left ventricular outflow tract gradient was technically inadequate.

**Conclusions:** Direct intraoperative measurement of pressures in the left ventricle and aorta provides important hemodynamic data in addition to intraoperative transesophageal echocardiography findings. This information assists the surgeon in defining the extent of myectomy. (J Thorac Cardiovasc Surg 2011;142:53-9)



Surgical myectomy is the preferred treatment for left ventricular (LV) outflow tract (LVOT) obstruction due to symptomatic hypertrophic obstructive cardiomyopathy (HOCM) when medical therapy proves unsuccessful.[1] LVOT obstruction in HOCM is a dynamic process. Changes in myocardial contractility, loading conditions, and heart rate substantially alter LVOT gradients.[2] The magnitude and potential impact of these changes on surgical decision-making within the operating room have not been well described. It is not uncommon for patients with severe symptomatic LVOT obstruction (> 30 mm Hg) to have a lower gradient under anesthesia than preoperatively; in contrast, patients with moderate LVOT gradients preoperatively may demonstrate severe obstruction in the operating room. LVOT obstruction can be ameliorated by anesthesia (eg, potent narcotics such as fentanyl induce bradycardia, and volatile anesthetics reduce contractility). LVOT obstruction also may be reduced by volume infusion and patient positioning (Trendelenburg) or, alternatively, it may be accentuated by decreased venous return due to anesthetic-induced vasodilation or hypovolemia common in patients who are fasting before surgery.

In this report we summarize our experience in the management of patients with HOCM, with an emphasis on the perioperative monitoring of the dynamics of LVOT obstruction.

## MATERIALS AND METHODS

### Study Patients

After institutional review board approval, we searched our clinic database for patients 18 years of age or older who had transaortic septal myectomy for symptomatic HOCM between 2004 and 2008. Of 549 patients identified, 198 consecutive patients had consented to participate in research and had medical records containing all the necessary information for the study: preoperative and intraoperative echocardiography reports, and scanned tracings of LV and aortic pressures measured directly in the operating room.

### Operative Technique

The standard surgical treatment of LVOT obstruction was transaortic septal myectomy.[3,4] The septum was exposed through an oblique

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**Abbreviations and Acronyms**

CPB = cardiopulmonary bypass
HOCM = hypertrophic obstructive cardiomyopathy
LV = left ventricle, left ventricular
LVOT = left ventricular outflow tract
MR = mitral regurgitation
MV = mitral valve
PVC = premature ventricular contraction
TEE = transesophageal echocardiography
TTE = transthoracic echocardiography

aortotomy, and an initial upward incision was made in the septal muscle at the nadir of the right aortic sinus. This incision was turned leftward to excise muscle over the anterior leaflet of the mitral valve (MV). The septal excision was deepened and lengthened toward the apex of the heart past the contact lesion (''scar'') on the endocardial surface. Operations were carried out with normothermic cardiopulmonary bypass (CPB), and hypothermic antegrade blood cardioplegia was used for myocardial protection.

## Anesthesia

Intraoperative management included standard monitoring (ie, electrocardiography, pulse oximetry, blood pressure cuff, direct arterial blood pressure, temperature), peripheral nerve stimulation, pulmonary artery catheterization, and transesophageal echocardiography (TEE).[5] Graphical trends of all principal hemodynamic parameters were displayed in real time and on the network computer at 1-minute intervals. For study purposes, we recorded hemodynamic parameters first after induction and then simultaneously with LVOT echocardiographic and direct measurements: (1) before myectomy 2 to 5 minutes before going on bypass; and (2) after myectomy 5 to 10 minutes postbypass after hemodynamic stabilization.

All patients had general anesthesia that consisted of administration of benzodiazepines (midazolam), opioids (fentanyl), volatile anesthetics (isoflurane), and muscle relaxants (pancuronium). We used calcium chloride post-CPB; if blood pressure was low after adequate volume replacement (mean arterial pressure, < 60 mm Hg), we administered vasoactive medications such as phenylephrine or vasopressin to restore normal systemic vascular resistance. Epinephrine was reserved for rare instances of poor cardiac performance and hypotension unresponsive to vasopressors. If atrioventricular block or bradycardia persisted after reperfusion, we used dual-chamber or atrial pacing. Electrolyte balance was controlled throughout surgery; we evaluated the concentration of potassium at least three times (postinduction, on CPB, and at closure) to maintain it within the reference range (3.6–5.2 mmol/L).

## LVOT Measurements

For echocardiographic evaluation of the LVOT obstruction, the transducer was positioned as parallel as possible to the LVOT jet to obtain maximal Doppler velocities. The Doppler-derived LVOT gradient was estimated at maximal velocity of blood flow through the LVOT during ventricular contraction (ie, the maximal instantaneous gradient) (Figure 1). Doppler velocity across the aortic valve was converted into a pressure gradient between the LV and the aorta by the modified Bernoulli equation ($\Delta p = 4v^2$). The Doppler maximal instantaneous gradient by definition was expected to be higher than the peak-to-peak gradient at the same point in the same cardiac cycle (Figure 1).

Patients with hypertrophic cardiomyopathy have an initial higher aortic pressure at very early systole due to unimpeded flow, which is then followed by a decrease in pressure and a gradual increase in pressure gradient. Thus, the maximal instantaneous gradient by Doppler echocardiography correlates best with the maximal gradient obtained by cardiac catheterization. Although this gradient may be the most accurate measure of the degree of obstruction, in clinical practice in the operating room, peak-to-peak systolic gradient is the easiest to obtain and provides reliable information with minimal underestimation of the true LVOT gradient.

Preoperatively, each of the 198 patients underwent comprehensive 2-dimensional and Doppler transthoracic echocardiography (TTE). Measurement of the LVOT gradient was carried out by continuous-wave Doppler interrogation of the LVOT from the apical window.

In the operating room, the grade of LVOT obstruction was evaluated twice: before myectomy 2 to 5 minutes before going on bypass and after

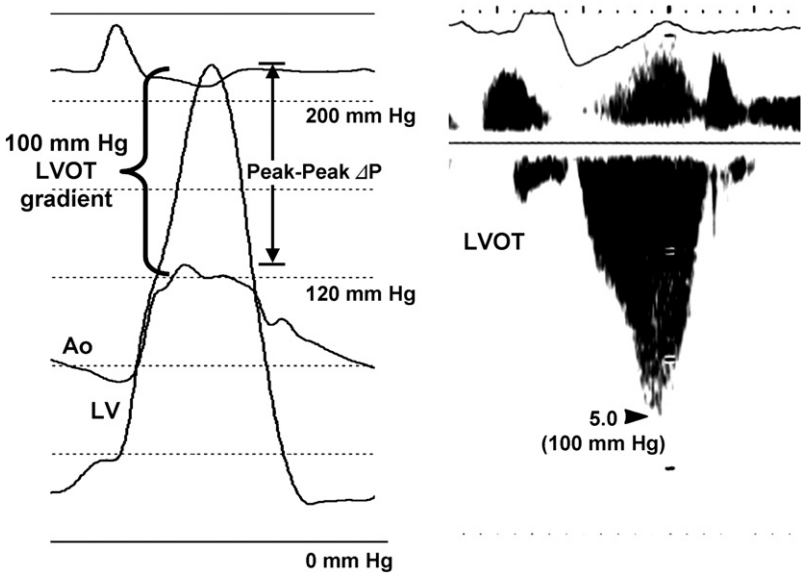


**FIGURE 1.** Maximal instantaneous gradient versus peak-to-peak gradient. Maximal instantaneous gradient (Max $\Delta$P) is calculated at the moment of maximal Ao (aortic) jet velocity ($V_{max}$) through the valve. Peak-to-peak gradient (Peak-Peak $\Delta$P) is calculated by subtraction of maximal left ventricular (LV) pressure from maximal Ao pressure.

myectomy 5 to 10 minutes postbypass after hemodynamic stabilization. LVOT flow was interrogated from the transgastric long-axis view by TEE. Direct measurement of the LVOT gradient was carried out by the operating surgeon, who inserted needles into the aorta near the cannulation site and into the LV through the right ventricle and septum; these were connected to separate fluid-filled lines and manometers. LV and aortic pressure tracings were recorded simultaneously, and the peak-to-peak gradient was calculated off-line by subtracting the peak systolic aortic pressure from the peak systolic LV pressure (Figures 2 and 3).

The LVOT gradient was first evaluated at baseline. In patients with low resting gradients (< 30 mm Hg), provocation maneuvers (eg, Valsalva or amyl nitrite inhalation) were applied preoperatively in the echocardiographic laboratory. The Valsalva maneuver consisted of breath suspension at the end of inspiration and straining down without breathing. For amyl nitrite provocation, the capsule was crushed and the patient inhaled its contents three times. In the operating room, provocation was by induction of premature ventricular contraction (PVC) or isoproterenol administration. PVC was induced by mechanical stimulation of the right ventricle. Isoproterenol challenge was carried out by titrated infusion via a pulmonary catheter, started at 1 $\mu$g/kg/min and increased at 3-minute intervals up to 4 $\mu$g/kg/min to achieve either a heart rate greater than 120 beats/min or an LVOT gradient greater than 50 mm Hg (Figure 2).

To minimize errors in evaluating Doppler velocity and calculating the directly measured gradient, we averaged three consecutive cardiac cycles if the patient had sinus or paced rhythm and five consecutive cardiac cycles for patients in atrial fibrillation.

### Statistical Analysis

Descriptive statistics for categorical variables were reported as frequency and percentage; continuous variables were expressed as mean ± standard deviation or median (range). Categorical variables were compared using the $\chi^2$ test. Continuous variables were compared using the 2-sample $t$ test or the Wilcoxon rank sum test. $P < .05$ was considered statistically significant.

## RESULTS

### Characteristics of Patients

The mean age of the patients was 52 ± 14 years, and the majority were men (Table 1). Generally, patients were overweight and had hyperdynamic and thickened LVs (LV ejection fraction, 72% ± 6%). The interventricular septal thickness was 21 ± 5 mm at end diastole, and the thickness of the posterior wall was 13 ± 3 mm, consistent with asymmetrical LV hypertrophy.

### Surgical Procedure

All 198 patients underwent transaortic septal myectomy, and 4 patients (2%) had an additional transapical incision for midventricular myectomy to relieve residual intracavitary obstruction. Twelve patients (6%) had concomitant MV repair (Table 1).

There was no perioperative mortality, and the operation was uncomplicated in 195 (98%) patients. Post-CPB TEE identified small iatrogenic ventricular septal defects in 2 patients, which were closed without a residual shunt. One patient had perforation of the LV free wall, subsequently repaired with bovine pericardium. This patient's initially unstable postoperative hemodynamics mandated insertion of an intra-aortic balloon pump, removed on postoperative day 3. The patient was discharged in stable condition 2 weeks after surgery.

After initial termination of CPB and direct measurement of intracardiac pressures, CPB was resumed in 19 (10%) patients. This decision was made because of a persistent

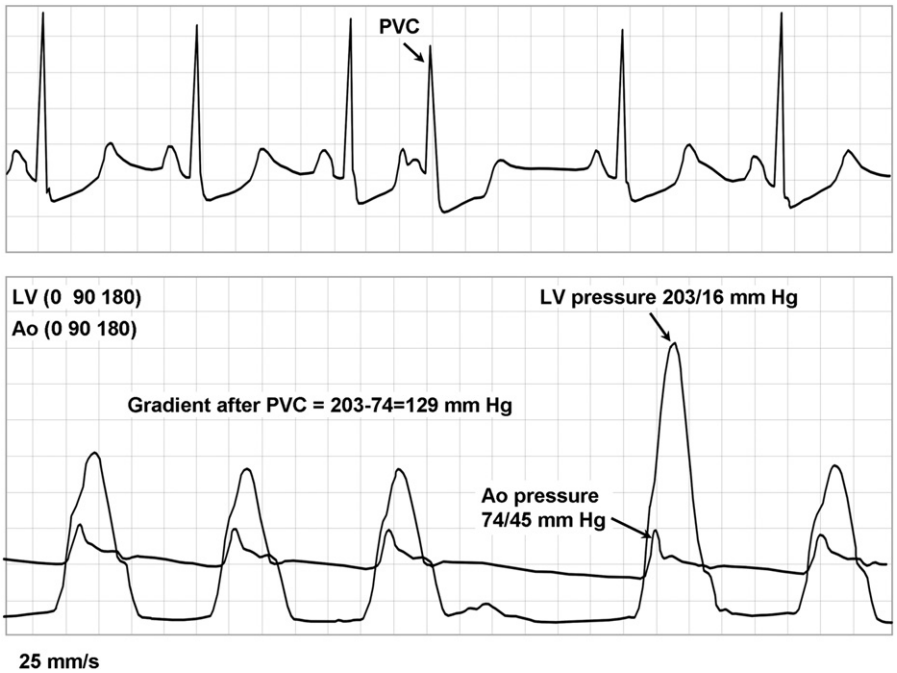


**FIGURE 2.** Peak-to-peak left ventricular outflow tract gradient after provocation test with premature ventricular contraction. Pressures in the left ventricle (LV) and in the aorta (Ao) are recorded simultaneously. Peak-to-peak gradient is calculated by subtraction of Ao pressure from LV pressure in systole. Note gradient increase after premature ventricular contraction (PVC).

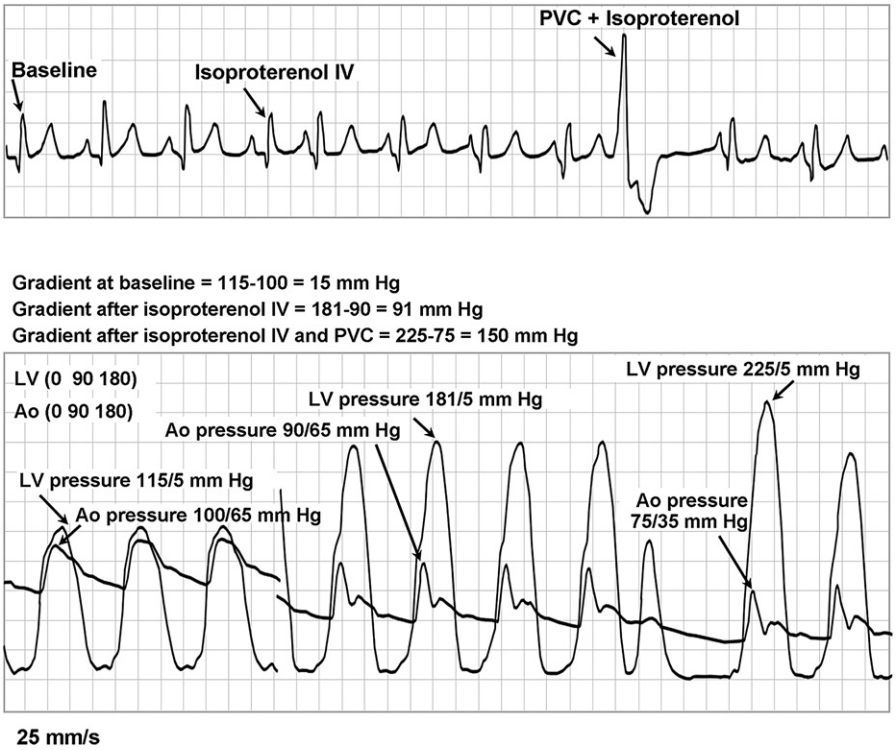


**FIGURE 3.** Left ventricular and aortic pressures before and after isoproterenol provocation. Note the increase of peak-to-peak left ventricular (LV) outflow tract gradient after isoproterenol infusion and subsequent premature ventricular contraction (PVC), compared with baseline measurements. *Ao*, Aortic; *IV*, intravenous.

residual LVOT gradient of 33 ± 14 mm Hg in 8 patients, moderately severe mitral regurgitation (MR) in 6 patients, and unstable hemodynamics in 1 patient. In the other 4 patients, the reasons for the second CPB were closure of iatrogenic ventricular septal defects (2 patients), removal of residual mobile tissue in the LV cavity revealed by TEE (1 patient), and interruption of ongoing bleeding from the left atrium suture site (1 patient).

## LVOT Gradients

Intraoperative hemodynamic data are summarized in Table 2. The baseline LVOT gradients were measured by TTE preoperatively and by direct measurements of left-sided intracardiac pressures intraoperatively (Table 3) in all 198 study patients. The baseline LVOT gradient was obtainable by intraoperative TEE in only 120 patients (61%). For 78 patients (39%), Doppler echocardiographic assessment of LVOT gradient could not be conducted because of the difficulty in aligning the Doppler beam parallel to the LVOT (Table 3). In 120 patients the average gradient measured before myectomy by direct measurement was similar to the gradient obtained by intraoperative TEE prebypass (52 ± 35 vs 58 ± 33 mm Hg, respectively; $P = .24$) (Table 3).

There were heterogeneous changes in LVOT obstruction when preoperative and intraoperative prebypass data were compared. After induction of anesthesia in 119 patients (60%), we observed milder LVOT obstruction (direct measurement) compared with preoperative values (TTE measurement): 41 ± 31 vs 76 ± 40 mm Hg ($P < .001$). In 75 patients (38%), the obstruction was more severe: 64 ± 32 vs 35 ± 31 mm Hg ($P < .001$); 4 patients (2%) had similar grades of LVOT obstruction before surgery and in the operating room (21 ± 27 mm Hg). On average, baseline

**TABLE 1. Baseline characteristics of patients undergoing septal transaortic myectomy**

| Characteristic | Value* (N = 198) |
|---|---|
| Age, y | 52 ± 14 |
| Male sex | 116 (59) |
| Body mass index, kg/m$^2$ | 31 ± 6 |
| Cardiopulmonary bypass time, min | 35 ± 19 |
| Cross-clamping time, min | 25 ± 15 |
| Concomitant procedures | |
| Mitral repair | 12 (6) |
| Coronary artery bypass graft | 10 (5) |
| Maze | 8 (4) |
| Aortic valve repair | 5 (3) |
| Foramen ovale closure | 4 (2) |
| Congenital VSD repair | 1 (1) |
| Preoperative ejection fraction, % | 72 ± 6 |

*VSD*, Ventricular septal defect. *Values are mean ± standard deviation or number (percentage) of patients.

**TABLE 2. Intraoperative hemodynamic data***

| Variable | Induction | Prebypass | *P* value† | Postbypass | *P* value‡ |
|---|---|---|---|---|---|
| Heart rate, beats/min | 69 ± 11 | 73 ± 15 | .001 | 85 ± 11 | < .001 |
| Heart rhythm | | | | | |
| Sinus | 182 (92) | 182 (92) | .99 | 127 (64) | < .001 |
| Atrial fibrillation | 7 (4) | 7 (4) | .99 | 1 (1) | .014 |
| Pacemaker | | | | | |
| Dual chamber | 6 (3) | 6 (3) | .99 | 37 (19) | < .001 |
| Atrial pacing | 3 (2) | 3 (2) | .99 | 33 (17) | < .001 |
| Blood pressure, mm Hg | | | | | |
| Systolic | 127 ± 23 | 121 ± 21 | .002 | 109 ± 13 | < .001 |
| Diastolic | 68 ± 14 | 68 ± 11 | .99 | 58 ± 9 | < .001 |
| Mean | 88 ± 16 | 86 ± 13 | .1 | 75 ± 9 | < .001 |
| Pulmonary artery pressure, mm Hg | | | | | |
| Systolic, mm Hg | 35 ± 11 | 34 ± 10 | .17 | 28 ± 7 | < .001 |
| Diastolic, mm Hg | 19 ± 7 | 18 ± 6 | .002 | 15 ± 5 | < .001 |
| Mean, mm Hg | 24 ± 8 | 23 ± 8 | .003 | 19 ± 6 | < .001 |
| RAP, mm Hg | 12 ± 5 | 10 ± 4 | < .001 | 9 ± 4 | < .001 |
| Cardiac output, L/min | | 4.9 ± 1.3 | | 5.3 ± 1.3 | < .001 |
| SVR, dyne × s/cm$^5$ | | 881 ± 358 | | 766 ± 296 | .001 |

*RAP*, Right atrial pressure; *SVR*, systemic vascular resistance. *Values are mean ± standard deviation or number (percentage) of patients. †*P* values are for comparison between the measurements at induction and at 2 to 5 minutes before going on bypass. ‡*P* values are for comparison between the measurements at induction and at 5 to 10 minutes postbypass after hemodynamic stabilization.

LVOT gradients measured directly intraoperatively were lower than resting gradients derived by preoperative TTE (52 ± 35 vs 69 ± 43 mm Hg; $P < .001$).

Provocation tests were used preoperatively in 100 patients (50%); many of these patients reported symptoms but had low (< 30 mm Hg) gradients at rest. Intraoperatively, we used provocation in all 198 patients. After provocation (PVC and/or isoproterenol) before myectomy, the grade of LVOT obstruction always increased substantially. Furthermore, LVOT gradients after provocation in the operating room were higher compared with those obtained preoperatively (Table 4).

We used intraoperative provocation with isoproterenol in 50 (25%) patients in this study when PVC failed to induce a sizable gradient. Isoproterenol administration increased the pressure gradient from 16 ± 22 to 75 ± 35 mm Hg ($P <$. 001); additional PVC induction increased the peak-to-peak gradient further to 95 ± 32 mm Hg ($P < .001$ vs isoproterenol alone) (Table 4).

**TABLE 3. Measurements of LVOT gradient at baseline (mm Hg)***

| Variable | Preoperative TTE | Intraoperative TEE | Intraoperative direct measurement |
|---|---|---|---|
| Before myectomy | | | |
| 198 patients | 59 ± 42 | | 49 ± 33 |
| 120 patients† | 69 ± 43 | 58 ± 33 | 52 ± 35 |
| After myectomy | | | |
| 198 patients | | 4 ± 6 | 4 ± 8 |

*LVOT*, Left ventricular outflow tract; *TEE*, transesophageal echocardiography; *TTE*, transthoracic echocardiography. *Values are mean ± standard deviation. †For 78 (39%) of the 198 patients, Doppler echocardiographic assessment of the LVOT gradient was technically inadequate (difficulty in aligning Doppler beam parallel to LVOT).

Septal myectomy decreased LVOT gradients in all 198 patients (Table 3). Residual gradients measured directly were generally less than 5 to 8 mm Hg at rest and less than 10 mm Hg with provocation. Residual postmyectomy LVOT gradient measured directly after provocation was 8 ± 10 vs 4 ± 6 mm Hg by TEE at rest (Table 3).

As mentioned above, in 8 patients (4%) the initial LVOT gradient after myectomy was greater than 25 mm Hg (average, 33 ± 14 mm Hg), prompting reinstitution of CPB for more extensive septal myectomy. The average gradient after repeat myectomy was 9 ± 9 mm Hg by direct measurement at baseline (or 6 ± 8 mm Hg by TEE) and 13 ± 7 mm Hg by direct measurement after PVC provocation.

## DISCUSSION

The dynamic pattern of LVOT obstruction has been appreciated since the 1960s,[6-9] and spontaneous daily fluctuations in the LVOT gradient of HOCM patients have

**TABLE 4. Measurements of LVOT gradient after provocation (mm Hg)***

| Provocation maneuver (patients [%]) | Baseline | Provocation | *P* value |
|---|---|---|---|
| Preoperative TTE | | | |
| Valsalva (94 [47]) | 36 ± 24 | 68 ± 32 | < .001 |
| Amyl nitrite (54 [27]) | 25 ± 20 | 82 ± 38 | < .001 |
| Intraoperative direct measurement | | | |
| PVC (169 [85]) | 49 ± 33 | 116 ± 53 | < .001 |
| Isoproterenol (38 [19]) | 16 ± 22 | 75 ± 35 | < .001 |
| Isoproterenol + PVC (12 [6]) | 18 ± 33 | 95 ± 32 | < .001 |

*LVOT*, Left ventricular outflow tract; *PVC*, premature ventricular contraction; *TTE*, transthoracic echocardiography. *Values are mean ± standard deviation.

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been described.[10] Intraoperatively, baseline hemodynamics may be altered compared with these in the conscious state because patients remain in the horizontal position at rest under anesthesia. Hypothetically, anesthetics could aggravate LVOT obstruction by decreasing blood pressure due to reduction of sympathetic tone. It is also important that all volatile anesthetics depress myocardial contractility, and this would tend to decrease the LVOT gradient.

In this study, we observed heterogeneous changes of LVOT gradients under anesthesia compared with preoperative measurements. On average, baseline LVOT gradients measured directly intraoperatively were lower compared with resting gradients derived by preoperative TTE. The estimated difference between mean values (17 mm Hg) might be due to negative inotropic effects of anesthesia; however, the peak-to-peak gradient measured directly is actually expected to be lower than the maximal instantaneous Doppler gradient (Figure 1).[11,12] Overall, 61% of study patients experienced a reduction of LVOT obstruction under anesthesia, but the rest of the study group had aggravation of LVOT obstruction. This variable response highlights the need to measure LVOT obstruction intraoperatively immediately before myectomy so that any residual postmyectomy gradient can be interpreted correctly.

In our experience, provocation tests can be helpful in unveiling the severity of LVOT obstruction in those who have mild gradients under anesthesia. Dynamic gradients can be provoked by several maneuvers. Preoperatively, the Valsalva maneuver was used most frequently in our patients. The maneuver increases intrathoracic pressure, thus decreasing venous return and reducing preload. Amyl nitrite is a potent vasodilator that also reduces preload and afterload, and this agent was used less frequently in our patients. Intraoperatively, LVOT gradients were provoked by induction of PVC (Brockenbrough–Braunwald–Morrow mechanism) and/or isoproterenol infusion, which increases contractility and reduces afterload.[6,13-15]

Echocardiography is an excellent diagnostic tool for detecting the presence of LVOT obstruction, and it also allows an estimation of the grade of associated MR and the presence of systolic anterior motion of the mitral leaflet. Unfortunately, in some cases it may be difficult to measure the LVOT gradient by intraoperative TEE. The Doppler beam must be aligned parallel to the maximal velocity vector, and it is important that the LVOT jet not be contaminated by the high-velocity MR signal when using continuous-wave Doppler.[16] For these reasons, Doppler echocardiographic assessment of the LVOT gradient was inadequate in 78 patients (39%) in this study. Thus, the information obtained by direct measurement of intracardiac pressures during myectomy is especially helpful.

In our current practice, resting and provoked LVOT gradients are measured directly in all patients both before and after myectomy. The accurate evaluation of any residual LVOT gradient after myectomy is critically important to assess the adequacy of the procedure. In this series, we resumed CPB in 8 patients (4%) to extend myectomy because of unacceptably high residual gradients; this rate of revision would be expected to be higher in practices that operate on fewer patients than our clinic handles (150 to 200 patients per year). With adequate myectomy at initial operation, the risk of late reoperation is very low (2%).[4] We believe that measurement of LVOT gradients is complementary to TEE and is especially helpful when reliable Doppler signals cannot be obtained.

### Study Limitations

There are several limitations of this study that should be acknowledged. First, the investigation was retrospective. Second, the study cohort included only 36% of patients undergoing myectomy during this interval. Excluded patients had incomplete data, but there is no reason to believe that these patients differed substantially from those under evaluation. The measurements of LVOT gradients and the Doppler echocardiograms were obtained in the course of clinical practice, and some measurements may not have been simultaneous.

## CONCLUSIONS

In patients with HOCM undergoing operation, anesthetics blunt LVOT obstruction in greater than 50%. For complete relief of LVOT obstruction, intraoperative hemodynamic monitoring consisting of TEE and direct measurement of provocable LVOT gradients is essential. This approach facilitates identification of any residual anatomical or functional (due to systolic anterior MV motion) LVOT obstruction. Assessment of the LVOT gradient by intraoperative TEE is not always technically feasible. The presence of residual LVOT gradients greater than 25 mm Hg should prompt consideration of resuming CPB for re-myectomy.

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